REMARKS

The undersigned would like to thank Examiner Myers for the courtesies extended during the interview conducted on March 4, 2002. The substance of the interview is recorded in form PTOL-413.

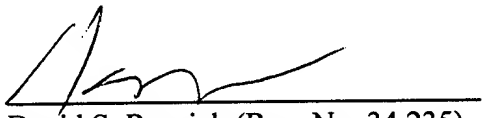
By the present Preliminary Amendment, Applicant has canceled claim 20 and added new claim 37. Support for new claim 37 can be found in claim 7 as originally filed. No new matter has been added by virtue of the amendment and entry of the new claim is respectfully requested.

In the event that there are any questions relating to this amendment or to the application in general, it would be appreciated if the Examiner would contact the undersigned attorney concerning such questions so that prosecution of this application can be expedited.

Entry of the foregoing and prompt and favorable consideration of the subject application on the merits are respectfully requested.

Date: 4/23/02

Respectfully submitted,



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VERSION WITH MARKINGS TO SHOW CHANGES MADE IN CLAIMS

13. A method of diagnosing a disease in a human subject comprising determining the presence of a genetic modification in a gene obtained from the subject which encodes a human G protein β_3 subunit.

14. The method as claimed in Claim 13, wherein said disease is a disorder associated with G protein dysregulation.

15. The method as claimed in Claim 13, wherein said gene which encodes a human G protein β_3 subunit has the nucleotide sequence of SEQ ID NO: 1.

16. The method as claimed in Claim 15, wherein the genetic modification is in the codon for amino acid 275 in SEQ ID NO: 1.

17. The method as claimed in Claim 16, wherein the genetic modification is a substitution of cytosine by thymine at position 825 in SEQ ID NO: 1.

18. The method as claimed in Claim 14, wherein the disorder is a cardiovascular disease, a metabolic disturbance or an immunological disease.

19. The method as claimed in Claim 14, wherein the disorder is hypertension.

20. (CANCELED)

21. (AMENDED) The method as claimed in Claim [20] 37, comprising comparing said gene obtained from a subject which encodes a human G protein β_3 subunit to the gene sequence of SEQ ID NO: 1.

22. The method as claimed in Claim 21, wherein the genetic modification which is determined is the presence of a thymine (T) at position 825 in the gene obtained from the subject.

23. (AMENDED) The method as claimed in Claim [20] 37, wherein the presence of a genetic modification in the gene obtained from a subject is determined by sequencing.

24. The method as claimed in Claim 23, further comprising the step of amplifying the gene obtained from the subject before sequencing.

25. The method as claimed in Claim 23, wherein a section the gene from the host corresponding to position 825 in the gene of SEQ ID NO: 1 is amplified.

26. (AMENDED) The method as claimed in Claim [20] 37, wherein the presence of a genetic modification in the gene obtained from the subject is determined by hybridization.

27. (AMENDED) The method as claimed in Claim [20] 37, wherein the presence of a genetic modification in the gene obtained from the subject is determined by cleavage using a restriction enzyme.

28. The method as claimed in Claim 27, wherein the restriction enzyme is Dsa I.

29. A non-human transgenic animal comprising a gene which encodes a modified human G protein β_3 subunit.

30. The non-human transgenic animal as claimed in Claim 29, which encodes a modified human G protein β_3 subunit of SEQ ID NO: 1.

31. The non-human transgenic animal as claimed in Claim 30, wherein said modified human G protein β_3 subunit includes a substitution of cytosine with thymine at position 825.

32. A method of diagnosing a disorder associated with G protein dysregulation, said method comprising:

(i) obtaining from a subject a gene which encodes a human G protein β_3 subunit;

(ii) determining the presence of a genetic modification in said gene from the nucleotide sequence of SEQ ID NO: 1; and

(iii) associating said genetic modification with said disorder.

33. The method as claimed in Claim 32 wherein said disorder is selected from the group consisting of cardiovascular disease, a metabolic disturbance, and an immunological disease.

34. The method as claimed in Claim 32 wherein said genetic modification in said gene is a substitution for cytosine by thymine at position 825 in SEQ ID NO: 1.

35. The method as claimed in Claim 32 wherein said subject is a human subject.

36. A method for diagnosing an increased likelihood of hypertension in a human subject comprising determining the presence of a genetic modification in a gene obtained from said subject which encodes a human G protein β_3 subunit by comparing said gene to the gene sequence of SEQ ID NO: 1, wherein said genetic modification is a substitution of cytosine by thymine at position 825 in SEQ ID NO: 1, wherein the presence of said genetic modification is associated with an increased likelihood of hypertension.

37. (NEW) A method for establishing a risk of developing disorders associated with G protein dysregulation for a subject, which comprises comparing the gene sequence for human G protein β3 subunit of the subject with the gene sequence SEQ ID NO:1, and , in the event that a thymine (T) is present at position 825, assigning the subject an increased risk of developing a disorder associated with G protein dysregulation.